World Neurosurgery: X 22 (2024) 100341

Contents lists available at ScienceDirect

World Neurosurgery: X

journal homepage: www.journals.elsevier.com/world-neurosurgery-x

Cognitive outcomes following aneurysmal subarachnoid hemorrhage: Rehabilitation strategies

Jihad Abdelgadir [a,*], Justin Gelman [b], Lindsay Dutko [c], Vikram Mehta [a], Allan Friedman [a], Ali Zomorodi [a]

[a] Department of Neurosurgery, Duke University Medical Center, Durham, NC, USA
[b] Trinity College of Arts and Sciences, Duke University, Durham, NC, USA
[c] Duke Speech Pathology, Duke University Medical Center, Durham, NC, USA

ARTICLE INFO

Keywords:
Cognitive outcomes
Neurorehabilitation
Subarachnoid hemorrhage

ABSTRACT

Despite decreases in mortality rate, the treatment of cognitive deficits following aneurysmal subarachnoid hemorrhage (aSAH) remains a serious challenge for clinicians and survivors alike. Deficits in executive function, language, and memory prevent more than half of survivors from returning to their previous level of work and put a tremendous amount of stress on the individual and their family. New therapies are needed for survivors of aSAH in order to improve cognitive outcomes and quality of life. The aim of this review is to discuss the prevalence and contributing factors of cognitive deficits following aSAH, as well as areas for therapeutic intervention. Due to the limited research on cognitive rehabilitative strategies for aSAH, a literature search of traumatic brain injury (TBI) was used to explore therapies with the potential to improve cognitive outcomes in aSAH. Across cognitive domains, existing rehabilitative and pharmacotherapeutic strategies for TBI show promise to be useful for survivors of aSAH. However, further study of these therapies in addition to consistent assessment of cognitive deficits are required to determine their efficacy in survivors of aSAH.

1. Introduction

Subarachnoid hemorrhage (SAH), most commonly caused by intracranial aneurysm rupture, is characterized by accumulation of blood in the subarachnoid space. The overall worldwide crude incidence of aneurysmal SAH (aSAH) was estimated to be 7.9 per 100,000 person-years, while the incidence in North America was estimated to be 6.9 per 100,000 person-years in 2010.[1] Some studies have suggested the incidence to be higher than documented due to misdiagnosis or death before arrival to the hospital.[2,3]

The majority of aSAH occur in patients between 40 and 60 years of age.[4,5] However, some studies report that children constitute a significant minority of cases, with aSAH accounting for more than 10% of childhood hemorrhagic strokes.[6]

Over the past 30 years, the mortality rate following aSAH has dropped significantly. Previously reported as high as ~48%,[7,8] advances in aSAH diagnosis and management have led to notably lower mortality rates in recent studies, with in-hospital mortality rates ranging from 8% to 24%.[7–10]

ASAH patients reportedly experience deficits in executive function, verbal and nonverbal memory, visual-spatial function psychomotor speed, and other cognitive domains.[11–13] Further, significant number of aSAH survivors report psychosocial and neurobehavioral changes that were disabling and burdensome to themselves and their family. Likewise, over 50% fail to return to the same level of work, which attests to the devastating nature of these deficits.[14,15] These cognitive deficits are not restricted to the immediate post-operative period. Survivors of aSAH experience accelerated cognitive decline at a greater rate than other neurovascular insults, including ischemic stroke, with a 2.6 times greater risk of dementia within 10 years of rupture compared to the general population.[16–18]

Even though the diagnosis and management of aSAH in the acute phase is well documented, the management of functional and cognitive deficits is not very well studied.[19] Research into health-related quality of life measures for aSAH patients with poor baseline cognitive function at discharge has demonstrated that quality of life outcomes are stratified by the presence or absence of improvement in cognitive deficits.[20] As such, optimizing cognitive function is both a crucial and potentially

* Corresponding author. Duke University Hospital DUMC Box 3271, Durham, NC 27710, USA.
E-mail address: jihad.abdelgadir@duke.edu (J. Abdelgadir).

https://doi.org/10.1016/j.wnsx.2024.100341
Received 13 September 2022; Accepted 21 February 2024
Available online 24 February 2024

intervenable area for improving the quality of life for aSAH patients. In this review, we aim to discuss the cognitive outcomes of aSAH and possible interventions for long term management.

2. Methods

There is very scarce data on rehabilitation strategies for aSAH survivors. Both traumatic brain injury (TBI) and aSAH include mechanical stress to brain tissue and an imbalance between cerebral blood flow, metabolism, excitotoxicity, edema formation, and inflammatory and apoptotic processes. Furthermore, the presentation and improvement of cognitive deficits in aSAH and TBI follow parallel pathways during the recovery process.[21] Due to similarities in cognitive outcomes and underlying pathophysiologic processes in aSAH and TBI, we thought it would be appropriate to extrapolate from the TBI literature on rehabilitation strategies that would be applicable to the aSAH population.

A literature review was conducted by searching PubMed for the key phrases" "Cognitive deficits", "Cognitive impairment", "Cognitive outcomes", and "Cognitive rehabilitation" with "intracranial bleeding", "stroke", and "subarachnoid hemorrhage".

3. Results and discussion

3.1. Cognitive outcomes

In most vascular brain injuries, sensory and motor deficits pose the biggest challenges. In aSAH however, cognitive impairment is the primary issue reported in survivors.[11] Predictably, there are numerous factors specific to patients and their clinical courses that affect the likelihood of global cognitive dysfunction after aSAH. One major predictor of poor cognitive outcomes is age, with patients under 50 demonstrating better neuropsychological function than their older counterparts.[22,23] After controlling for age, factors such as treatment method and presence of delayed cerebral ischemia (DCI) due to cerebral vasospasm may also play a role. For example, microsurgical clipping is associated with cognitive impairment compared to endovascular coiling in the immediate postoperative period and at 12-months post--intervention.[24,25] While the effect of aneurysm location on mean neuropsychological performance is equivocal, there may be an association between aneurysm location and particular cognitive domains.[23] Yet, there is an association between the distribution of DCI and cognitive outcomes, with infarcts due to vasospasm of the middle cerebral artery and its perforators associated with worse performances on the Montreal Cognitive Assessment and Mini-Mental Status Exam at 3-months.[23]

Nonetheless, it is more useful to describe cognitive outcomes in terms of deficits in specific cognitive domains, as different forms of dysfunction require different treatment approaches. Outcomes can be further described by specific domains of cognitive function. In this context, over 70% of aSAH survivors are found to have deficits in attention, executive function, memory, language and mood.[26,27]

3.2. Executive function

Executive function is a term used to encapsulate a broad-range of hypothesized cognitive processes, including planning, working memory, attention, inhibition, self-monitoring, self-regulation, and initiation.[28] It is thought to be predominantly mediated by prefrontal areas of the frontal lobe.[28,29] Given the diversity of cognitive processes considered executive function and the variation in neuropsychological tests used in studies, the prevalence of executive dysfunction in survivors of aSAH broadly ranges from approximately 3%–75%.[12,30,31] Kreiter and colleagues reported that age and education level are significant predictors of executive dysfunction after aSAH, as deficits are more pronounced in older patients with fewer years of education.[32] Hunt and Hess grade was not found to be highly predictive of deficits in executive function, suggesting that secondary insults like hydrocephalus and complications of ischemia may play a more important role than the initial injury.[13,32] However, the impact of aneurysm location on the degree of cognitive dysfunction is inconclusive. While some studies reported anterior communicating artery, left anterior circulation, and vertebrobasilar system aneurysms to be associated with poor cognitive outcome,[33–35] others reported no relationship between aneurysm location and cognitive impairment.[36–39] The main culprit of cognitive dysfunction has been reported to be the contact of subarachnoid blood with basal frontal and perisylvian cortices.[11,32] In the same study, Kreiter and colleagues found that filling of the sylvian and anterior interhemispheric fissures with dense subarachnoid blood was strongly associated with dysfunction in visual and verbal memory.[32] On the contrary, other investigators hypothesized that rather than focal lesions, aSAH causes global damage to the brain through elevated intracranial pressure, reduced blood flow, breakdown of the blood brain barrier, and global cerebral edema: the diffuse damage hypothesis.[40,41] Bendel and colleagues' findings on structural changes to the brains of aSAH survivors supported this hypothesis. After 1-year post-rupture, aSAH survivors showed ventricular and sulcal enlargement as well as significantly reduced total gray and white matter volume compared to the average population. Increasing gray matter volume loss was associated with poorer executive function.[42]

Table 1
Prevalence and factors correlated with impairment across cognitive domains.

Cognitive Domain	Prevalence	Age	Education	Neurological Grade	Circulation
Executive function	3%–75%	+	–	NA	Inconclusive
Language	0%–76%	+	–	+	Anterior
Memory	14%–61%	+	–	+	Anterior

+ = positive correlation with cognitive deficit, – = negative correlation with cognitive deficit, NA = no association.

The prevalence of executive dysfunction is affected by the length of the follow up period. Several investigators found that some domains of executive function significantly improve within the first year after hospital discharge.[43,44] However, a study by Haug and colleagues suggests that different aspects of executive function recover at different rates.[39] Although deficits in inhibition improved within 1-year after aSAH, impairments in cognitive flexibility and attention showed no improvement over the same period. These findings from longitudinal studies must be interpreted cautiously because tests of executive function may be subject to practice effects.[45,46] In order to accurately assess executive function in aSAH survivors, specific domains of executive function as well as follow up length and interval should be carefully considered.

3.3. Language

Language function is a broad category that encompasses the comprehension and expression of meaningful written and oral information. Similar to executive function, the prevalence of language impairment reported in aSAH survivors is quite variable. Due to the broad categories and different assessments used to define language dysfunction, studies report language deficits ranging from 0 % to 76% (Table 1).[12,31] Factors including age, lack of education and anterior circulation aneurysms have also been linked worsening language function in aSAH.[32]

Yet, studies have shown significant improvements in language function within the first three months after aSAH that continue until 18 months post-rupture.[47] This improvement is not subject to practice effect, given the nature of tests used for assessment.

Table 2
Examples of standardized neuropsychological assessment tests.

Cognitive Domain	Standard assessment tests
Executive function	Stroop test, Trail Making Test, Wisconsin Card Sorting Test, Conners' Continuous Performance Test-II (CCPT-II), Paced Auditory Serial Attention Test (PASAT)
Language	Boston Naming Test, Multilingual Aphasia Examination Token Test, Controlled Oral Word Association Test
Memory	Continuous Visual Memory Test (CVMT), Continuous Verbal Memory Test (CVLT), Wechsler Adult Intelligence Scale III (WAIS-III)

3.4. Memory

ASAH survivors suffer memory impairments that affect verbal, visual, short-term, and long-term memory. Deficits in verbal memory are most common with a prevalence of 14%–61%.[12,31] The prevalence of memory impairment in aSAH survivors depends on the type of memory in question, tests used and time of testing.[47] Similar to improvements in language dysfunction, improvements in verbal and visual memory have been shown over time.[47] Additionally, factors such as age, level of education, neurological grade on admission and anterior circulation aneurysm have been reported to be significantly associated with memory impairment in aSAH survivors.[32]

3.5. Cognitive assessment

In order to plan cognitive rehabilitation for aSAH survivors, detailed neuropsychiatric assessment is critical to assess cognitive abilities and identify cognitive deficits. Additionally, repeating these assessments at regular intervals is crucial to evaluate the effectiveness of ongoing treatment. Table 2 below shows some of the commonly used standard assessment tests for executive function, language, and memory.

However, progress in neuropsychiatric assessments do not necessarily translate to improvements in functional outcomes and ability to perform activities of daily living (ADLs). Therefore, the functional independence measure (FIM) and Disability Rating Scale (DRS) to assess functional outcomes should be employed in conjunction with neuropsychiatric assessments to plan rehabilitation programs and evaluate the effectiveness of ongoing treatment.

There is strong evidence that attention can be improved with specific skills training in patients with acquired brain injuries like TBI or aSAH.[48,49] Attention process training (APT) is a direct attention training program that has been designed to improve and hopefully restore visual and auditory attention.[49,50] APT targets five components of attention: Focused attention, sustained attention, selective attention, alternating attention, and divided attention. The training program consists of tasks with a hierarchical progression of increasing attention demands that progress from simple to complex distracters. APT can significantly improve complex attention, as TBI and stroke patients undergoing APT performed better in the Paced Auditory Serial Addition Test in multiple studies.[49,51] Metacognitive remediation and cognitive behavioral psychotherapy are also shown to improve attention and overall executive function.[52] Specifically, these therapies facilitate the treatment of attention, memory, language deficits, and social skills.[53] Metacognitive training improves deficits in executive function by targeting the development of compensatory strategies. Through structured and repetitive cueing or encouraging repeated assessment and self-monitoring, metacognitive training helps to assess individual performance and reduces or prevents errors.[54] Cognitive remediation alternatively includes direct attention training and compensatory strategy training with memory notebook and problem-solving strategies. Additionally, problem-solving training (PST) and goal management training have also been shown to be beneficial in improving executive function in persons with acquired brain injury.[55,56]

3.6. Pharmacotherapy

Amantadine, a dopamine receptor agonist, may facilitate the recovery of the nervous system after acquired brain injury. Amantadine is also an NMDA receptor antagonist, protecting neural cells against excitotoxicity. There is strong evidence to support the use of amantadine for cognitive rehabilitation in the acute phase of TBI.[57,58] For example, a randomized controlled trial of 184 patients in unresponsive wakefulness syndrome or a minimally conscious state at 4–16 weeks post injury assigned participants to receive amantadine or placebo for 4 weeks.[56] When administered within the first few days following injury, the results supported that amantadine enhances attention, arousal, visuospatial function and overall executive function in TBI patients.[57] However, it's efficacy in chronic TBI is questionable. Another randomized controlled trial found that amantadine does not have an impact on cognition in chronic TBI patients beyond 6 months post-injury.[59] Another challenge is that dosing and duration of amantadine use remain unclear. A recent study in rats with cortical injury found that amantadine (20 mg/kg) improved beam-balance recovery and spatial learning relative to placebo.[60] In the same study, no other doses were found to be effective. This indicates that the proper dosing of amantadine is crucial to its effect on cognitive recovery and should be carefully considered.

Bromocriptine, a D2 dopamine receptor agonist, has also shown promise in mitigating deficits in executive function. A double-blind, placebo-controlled crossover trial tested a low dose of bromocriptine (2.5 mg/day) on 24 subjects. They found that there were gains in dual-task performance and on clinical measures of executive function.[61]

3.7. Language

Given the complexity of language disorders, we will discuss aphasia, apraxia, and social communication deficits for the purpose of this review. Speech and language therapy have been found to improve dysarthria and aphasia in acquired brain injuries. Some of the speech and language therapy strategies include constraint-induced aphasia therapy (CIAT).[62–64] CIAT relies on the principle of continuous practice and uses language tasks of increasing difficulty with the constraint of compensatory (nonverbal) communication strategies.[63] For those with apraxia - the inability to carry out a motor act despite intact motor and sensory pathways - Smania and colleagues found that gesture production exercises significantly improved movement deficits.[65]

Impairments in social communication affect the vast majority of patients with TBI and significantly impact their ability to interact with others.[66] Two randomized controlled trials have shown that pragmatic language skills, social behaviors, and cognitive training, along with psychotherapy for emotional adjustment, can significantly improve social communication skills of acquired brain injury persons.[67,68] Other methods that are shown to improve social communication skills of persons who suffered a brain injury are group-based interventions and specialized computer and internet training material.[69,70]

An effective intervention for improving conversation skills among individuals with TBI is communication partner training.[71] In particular, the TBIConneCT program trains both individuals with TBI and their common conversational partners to increase collaboration and decrease maladaptive behaviors thus creating more opportunities for effective conversation.[72] After the TBIConneCT partner training, a recent study demonstrated conversation abilities improved for both TBI and non-TBI participants according to scores on the Measure of Participation in Conversation and the Measure of Support in Conversation rating scales as well as a blinded assessor.[72]

3.8. Memory

Cognitive rehabilitation therapies focus on either restoration or compensation for memory deficits.[13] Thus far, there is no strong data to support the efficacy of restorative strategies.[53,70]

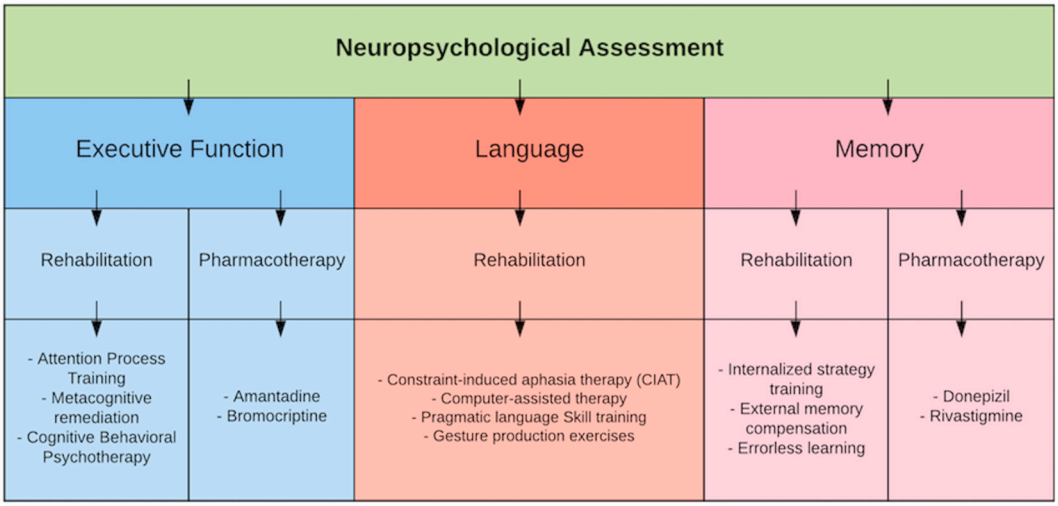

Fig. 1. Flow-chart summarizing rehabilitation strategies and pharmacological interventions to address deficits in executive function, language, and memory after TBI.

As for compensatory strategies, there is strong evidence supporting the use of external memory aids in compensating for memory impairments. Compensatory strategy training includes internalized strategy training, such as visual imagery, and external memory compensations, such as memory notebooks and assistive technology tools. These strategies were found to be helpful in memory impairments after TBI and may be effective in aSAH as well.[70]

Errorless learning, a method that facilitates compensatory strategies training by targeting individual-specific memory problems, also shows promise to be useful in memory impairments.[70,73,74] In a randomized study to evaluate the effectiveness of a computerized, errorless, learning-based memory rehabilitation program, Dou and colleagues showed that the combined use of these approaches may be an effective way of enhancing memory in persons with acquired brain injury.[73]

3.9. Pharmacotherapy

Multiple studies have shown that donepezil, a centrally selective acetylcholinesterase inhibitor, can improve attention and memory impairments in patients with acquired brain injury.[75,76] Additionally, a 12-week course of rivastigmine, acetylcholinesterase and butyrylcholinesterase inhibitor demonstrated a positive impact on attention and working memory in aSAH survivors and TBI patients at a dosage reported for rivastigmine is 3–6 mg/day.[77,78]

4. Discussion

As the field of neurorehabilitation in aSAH continues to evolve, we present the first review to use the link between cognitive outcomes in TBI and aSAH as a launching point to explore future directions for improving cognitive deficits aSAH. While there is a paucity of randomized trials and literature in general that discusses ways to improve cognitive dysfunction in aSAH patients, the rich literature in TBI should be utilized to improve cognitive outcomes in aSAH as covered in this review. This paradigm shift is important to hasten the development of new protocols to address cognitive outcomes in aSAH survivors, which is a relatively smaller population with less funded research than the TBI population.

First and foremost, aSAH patients require rigorous neuropsychological and functional evaluation before patients leave the hospital. It is crucial that domain-specific assessments in executive function, language, and memory are given to determine the particular deficits challenging patients on an individualized basis. By engaging in early and frequent assessment, specific intervention plans can be created and, depending on their condition, even be employed before the patient leaves the hospital. Overall, this highlights the need for strong communication between hospital providers and rehabilitation facilities to effectively coordinate care for patients.

After identifying cognitive deficits, the interventions reviewed may provide benefits to survivors of aSAH (Fig. 1). For instance, APT, metacognitive training, and PST, as well as potential pharmacological interventions like bromocriptine and amantadine, may improve deficiencies in areas of executive function. For language deficits, CIAT and other group-based communication interventions are promising treatments for survivors of aSAH. Finally, compensatory strategies like errorless learning and internalized strategy training, in addition to pharmacological treatments like donepezil and rivastigmine, should be explored to address memory issues.

5. Limitations

While TBI and aSAH do share many characteristics that warrant comparison, they are nonetheless distinct clinical entities that may impact cognitive outcomes. These differences include but are not limited to the injuries distinct to TBI such as coup-contrecoup injuries and non-SAH bleeds, as well as difficulty quantifying the true prevalence of vasospasm and DCI in TBI (Fawaz et al 2017). As a result, further research is needed to demonstrate that the strategies currently employed to support cognitive recovery in the TBI population are similarly effective for survivors of aSAH. Additionally, this review was not systematic and thus there may be additional rehabilitative strategies not covered in this review that warrant further exploration.

6. Conclusion

Given the similarities between TBI and aSAH, the interventions shown to be efficacious in improving cognitive deficits in TBI warrant further exploration in aSAH. In order to get more conclusive answers on the efficacy of these treatments for survivors of aSAH, controlled studies paired with standardized and frequent neuropsychological assessment of patients are required. Furthermore, understanding the impact of treatment onset and duration as well as the sustainability of cognitive improvements is crucial in determining the ideal approach to address cognitive deficits. Nonetheless, the TBI literature can hopefully serve as the foundation for mitigating the devastating cognitive deficits seen in survivors of aSAH.

CRediT authorship contribution statement

Jihad Abdelgadir: Conceptualization, Investigation, Formal analysis, Writing–original draft. **Justin Gelman:** Investigation, Writing–original draft, Writing–review and editing. **Linsday Dutko:** Writing–review and editing. **Vikram Mehta:** Writing–review and editing. **Allan Friedman:** Conceptualization, Supervision, Writing–review and editing. **Ali Zomorodi:** Conceptualization, Supervision, Writing–review and editing.

Declaration of competing interest

None

Disclosure/Acknowledgements

No disclosures or acknowledgements.

References

1. Etminan N, Chang H-S, Hackenberg K, et al. Worldwide incidence of aneurysmal subarachnoid hemorrhage according to region, time period, blood pressure, and smoking prevalence in the population: a systematic review and meta-analysis. *JAMA Neurol*. 2019;76(5):588–597.
2. Polmear A. Sentinel headaches in aneurysmal subarachnoid haemorrhage: what is the true incidence? A systematic review. *Cephalalgia*. 2003;23(10):935–941.
3. Kowalski RG, Claassen J, Kreiter KT, et al. Initial misdiagnosis and outcome after subarachnoid hemorrhage. *JAMA*. 2004;291(7):866–869.
4. Rinkel GJ, Djibuti M, Algra A, Van Gijn J. Prevalence and risk of rupture of intracranial aneurysms: a systematic review. *Stroke*. 1998;29(1):251–256.
5. Ingall T, Asplund K, Mähönen M, Bonita R. A multinational comparison of subarachnoid hemorrhage epidemiology in the WHO MONICA stroke study. *Stroke*. 2000;31(5):1054–1061.
6. Jordan LC, Johnston SC, Wu YW, Sidney S, Fullerton HJ. The importance of cerebral aneurysms in childhood hemorrhagic stroke: a population-based study. *Stroke*. 2009;40(2):400–405.
7. Broderick JP, Brott T, Tomsick T, Miller R, Huster G. Intracerebral hemorrhage more than twice as common as subarachnoid hemorrhage. *J Neurosurg*. 1993;78(2):188–191.
8. Sarti C, Tuomilehto J, Salomaa V, et al. Epidemiology of subarachnoid hemorrhage in Finland from 1983 to 1985. *Stroke*. 1991;22(7):848–853.
9. Molyneux AJ, Kerr RS, Birks J, et al. Risk of recurrent subarachnoid haemorrhage, death, or dependence and standardised mortality ratios after clipping or coiling of an intracranial aneurysm in the International Subarachnoid Aneurysm Trial (ISAT): long-term follow-up. *Lancet Neurol*. 2009;8(5):427–433.
10. Andaluz N, Zuccarello M. Recent trends in the treatment of cerebral aneurysms: analysis of a nationwide inpatient database. *J Neurosurg*. 2008;108(6):1163–1169.
11. Ogden JA, Mee EW, Henning M. A prospective study of impairment of cognition and memory and recovery after subarachnoid hemorrhage. *Neurosurgery*. 1993;33(4):572–587.
12. Mayer S, Kreiter K, Copeland D, et al. Global and domain-specific cognitive impairment and outcome after subarachnoid hemorrhage. *Neurology*. 2002;59(11):1750–1758.
13. Hadjivassiliou M, Tooth C, Romanowski C, et al. Aneurysmal SAH: cognitive outcome and structural damage after clipping or coiling. *Neurology*. 2001;56(12):1672–1677.
14. Buchanan KM, Elias LJ, Goplen GB. Differing perspectives on outcome after subarachnoid hemorrhage: the patient, the relative, the neurosurgeon. *Neurosurgery*. 2000;46(4):831–840.
15. Lindberg M, Angquist K-A, Fodstad H, Fugl-Meyer K, Fugl-Meyer AR. Self-reported prevalence of disability after subarachnoid haemorrhage, with special emphasis on return to leisure and work. *Br J Neurosurg*. 1992;6(4):297–304.
16. Wong GKC, Nung RCH, Sitt JCM, et al. Location, infarct load, and 3-month outcomes of delayed cerebral infarction after aneurysmal subarachnoid hemorrhage. *Stroke*. 2015;46(11):3099–3104. https://doi.org/10.1161/STROKEAHA.115.010844.
17. Rost NS, Brodtmann A, Pase MP, et al. Post-stroke cognitive impairment and dementia. *Circ Res*. 2022;130(8):1252–1271.
18. Corraini P, Henderson VW, Ording AG, Pedersen L, Horváth-Puhó E, Sørensen HT. Long-term risk of dementia among survivors of ischemic or hemorrhagic stroke. *Stroke*. 2017;48:180–186, 10.1161/STROKEAHA.116.015242.
19. Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. *N Engl J Med*. 2006;354(4):387–396.
20. Winter Y, Klotsche J, Ringel F, et al. Characterizing the individual course of health-related quality of life after subarachnoid haemorrhage: latent growth mixture modelling. *J Stroke Cerebrovasc Dis*. 2023;32(3):106913.
21. Tölli A, Höybye C, Bellander BM, Johansson F, Borg J. The effect of time on cognitive impairments after non-traumatic subarachnoid haemorrhage and after traumatic brain injury. *Brain Inj*. 2018;32(12):1465–1476.
22. Springer MV, Schmidt JM, Wartenberg KE, Frontera JA, Badjatia N, Mayer SA. Predictors of global cognitive impairment 1 year after subarachnoid hemorrhage. *Neurosurgery*. 2009;65(6):1043–1051. https://doi.org/10.1227/01.NEU.0000359317.15269.20.
23. Orbo M, Waterloo K, Egge A, Isaksen J, Ingebrigtsen T, Romner B. Predictors for cognitive impairment one year after surgery for aneurysmal subarachnoid hemorrhage. *J Neurol*. 2008 Nov;255(11):1770–1776. https://doi.org/10.1007/s00415-008-0047-z. Epub 2008 Oct 7. PMID: 18854912.
24. Vieira ACC, Azevedo-Filho HR, Andrade G, et al. Cognitive changes in patients with aneurysmal subarachnoid hemorrhage before and early posttreatment: differences between surgical and endovascular. *World Neurosurgery*. 2012;78(1):95–100.
25. Scott RB, Eccles F, Molyneux AJ, Kerr RSC, Rothwell PM, Carpenter K. Improved cognitive outcomes with endovascular coiling of ruptured intracranial aneurysms neuropsychological outcomes from the international subarachnoid aneurysm trial (ISAT). *Stroke*. 2010;41(8):1743–1747. https://doi.org/10.1161/STROKEAHA.110.585240.
26. Visser-Meily JA, Rhebergen ML, Rinkel GJ, van Zandvoort MJ, Post MW. Long-term health-related quality of life after aneurysmal subarachnoid hemorrhage: relationship with psychological symptoms and personality characteristics. *Stroke*. 2009;40(4):1526–1529.
27. Vetkas A, Lepik T, Eilat T, Rätsep T, Asser T. Emotional health and quality of life after aneurysmal subarachnoid hemorrhage. *Acta Neurochir*. 2013;155(6):1107–1114.
28. Otero TM, Barker LA. The frontal lobes and executive functioning. In: *Handbook of Executive Functioning*. Springer; 2014:29–44.
29. Stuss DT, Levine B. Adult clinical neuropsychology: lessons from studies of the frontal lobes. *Annu Rev Psychol*. 2002;53(1):401–433.
30. Mavaddat N, Sahakian BJ, Hutchinson PJ, Kirkpatrick PJ. Cognition following subarachnoid hemorrhage from anterior communicating artery aneurysm: relation to timing of surgery. *J Neurosurg*. 1999;91(3):402–407.
31. Ørbo M, Waterloo K, Egge A, Isaksen J, Ingebrigtsen T, Romner B. Predictors for cognitive impairment one year after surgery for aneurysmal subarachnoid hemorrhage. *J Neurol*. 2008;255(11):1770–1776.
32. Kreiter KT, Copeland D, Bernardini GL, et al. Predictors of cognitive dysfunction after subarachnoid hemorrhage. *Stroke*. 2002;33(1):200–208.
33. Stabell KE, Magnæs B. Neuropsychological course after surgery for intracranial aneurysms. A prospective study and a critical review. *Scand J Psychol*. 1997;38(2):127–137.
34. Richardson J. Cognitive performance following rupture and repair of intracranial aneurysm. *Acta Neurol Scand*. 1991;83(2):110–122.
35. Bornstein R, Weir B, Petruk K, Disney L. Neuropsychological function in patients after subarachnoid hemorrhage. *Neurosurgery*. 1987;21(5):651–654.
36. Hillis AE, Anderson N, Sampath P, Rigamonti D. Cognitive impairments after surgical repair of ruptured and unruptured aneurysms. *J Neurol Neurosurg Psychiatry*. 2000;69(5):608–615.
37. Egge A, Waterloo K, Sjøholm H, et al. Outcome 1 year after aneurysmal subarachnoid hemorrhage: relation between cognitive performance and neuroimaging. *Acta Neurol Scand*. 2005;112(2):76–80.
38. Samra SK, Giordani B, Caveney AF, et al. Recovery of cognitive function after surgery for aneurysmal subarachnoid hemorrhage. *Stroke*. 2007;38(6):1864–1872.
39. Haug T, Sorteberg A, Sorteberg W, Lindegaard K-F, Lundar T, Finset A. Cognitive outcome after aneurysmal subarachnoid hemorrhage: time course of recovery and relationship to clinical, radiological, and management parameters. *Neurosurgery*. 2007;60(4):649–657.
40. Uski TK, Lilja Å, Säveland H, Ekman R, Sonesson B, Brandt L. Cognitive functioning and cerebrospinal fluid concentrations of neuropeptides for patients with good neurological outcomes after aneurysmal subarachnoid hemorrhage. *Neurosurgery*. 2000;47(4):812–818.
41. Cahill J, Zhang JH. Subarachnoid hemorrhage: is it time for a new direction? *Stroke*. 2009;40(3_suppl_1):S86–S87.
42. Bendel P, Koivisto T, Äikiä M, et al. Atrophic enlargement of CSF volume after subarachnoid hemorrhage: correlation with neuropsychological outcome. *Am J Neuroradiol*. 2010;31(2):370–376.
43. Koivisto T, Vanninen R, Hurskainen H, Saari T, Hernesniemi J, Vapalahti M. Outcomes of early endovascular versus surgical treatment of ruptured cerebral aneurysms: a prospective randomized study. *Stroke*. 2000;31(10):2369–2377.
44. Frazer D, Ahuja A, Watkins L, Cipolotti L. Coiling versus clipping for the treatment of aneurysmal subarachnoid hemorrhage: a longitudinal investigation into cognitive outcome. *Neurosurgery*. 2007;60(3):434–442.
45. Dikmen SS, Heaton RK, Grant I, Temkin NR. Test–retest reliability and practice effects of expanded halstead–reitan neuropsychological test battery. *J Int Neuropsychol Soc*. 1999;5(4):346–356.
46. Basso MR, Bornstein RA, Lang JM. Practice effects on commonly used measures of executive function across twelve months. *Clin Neuropsychol*. 1999;13(3):283–292.
47. Powell J, Kitchen N, Heslin J, Greenwood R. Psychosocial outcomes at 18 months after good neurological recovery from aneurysmal subarachnoid haemorrhage. *J Neurol Neurosurg Psychiatry*. 2004;75(8):1119–1124.
48. Park NW, Ingles JL. Effectiveness of attention rehabilitation after an acquired brain injury: a meta-analysis. *Neuropsychology*. 2001;15(2):199.
49. Barker-Collo SL, Feigin VL, Lawes CM, Parag V, Senior H, Rodgers A. Reducing attention deficits after stroke using attention process training: a randomized controlled trial. *Stroke*. 2009;40(10):3293–3298.
50. Moore Sohlberg M, McLaughlin KA, Pavese A, Heidrich A, Posner MI. Evaluation of attention process training and brain injury education in persons with acquired brain injury. *J Clin Exp Neuropsychol*. 2000;22(5):656–676.
51. Sohlberg MM, Mateer CA. Effectiveness of an attention-training program. *J Clin Exp Neuropsychol*. 1987;9(2):117–130.

52. Tiersky LA, Anselmi V, Johnston MV, et al. A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Arch Phys Med Rehabil.* 2005;86(8):1565–1574.
53. Cicerone KD, Dahlberg C, Malec JF, et al. Evidence-based cognitive rehabilitation: updated review of the literature from 1998 through 2002. *Arch Phys Med Rehabil.* 2005;86(8):1681–1692.
54. Goverover Y, Johnston MV, Toglia J, DeLuca J. Treatment to improve self-awareness in persons with acquired brain injury. *Brain Inj*. 2007;21(9):913–923.
55. von Cramon DY, Cramon GM, Mai N. Problem-solving deficits in brain-injured patients: a therapeutic approach. *Neuropsychol Rehabil.* 1991;1(1):45–64.
56. Levine B, Robertson IH, Clare L, et al. Rehabilitation of executive functioning: an experimental–clinical validation of Goal Management Training. *J Int Neuropsychol Soc*. 2000;6(3):299–312.
57. Giacino JT, Whyte J, Bagiella E, et al. Placebo-controlled trial of amantadine for severe traumatic brain injury. *N Engl J Med.* 2012;366(9):819–826.
58. Kraus MF, Maki PM. Effect of amantadine hydrochloride on symptoms of frontal lobe dysfunction in brain injury: case studies and review. *J Neuropsychiatry Clin Neurosci.* 1997;9(2):222–230.
59. Hammond FM, Sherer M, Malec JF, et al. Amantadine did not positively impact cognition in chronic traumatic brain injury: a multi-site, randomized, controlled trial. *J Neurotrauma*. 2018;35(19):2298–2305.
60. Okigbo AA, Helkowski MS, Royes BJ, et al. Dose-dependent neurorestorative effects of amantadine after cortical impact injury. *Neurosci Lett.* 2019;694:69–73.
61. McDowell S, Whyte J, D'Esposito M. Differential effect of a dopaminergic agonist on prefrontal function in traumatic brain injury patients. *Brain J Neurol*. 1998;121(6):1155–1164.
62. Sickert A, Anders L-C, Münte TF, Sailer M. Constraint-induced aphasia therapy following sub-acute stroke: a single-blind, randomised clinical trial of a modified therapy schedule. *J Neurol Neurosurg Psychiatry*. 2014;85(1):51–55.
63. Meinzer M, Djundja D, Barthel G, Elbert T, Rockstroh B. Long-term stability of improved language functions in chronic aphasia after constraint-induced aphasia therapy. *Stroke*. 2005;36(7):1462–1466.
64. Raymer AM, Kohen FP, Saffell D. Computerised training for impairments of word comprehension and retrieval in aphasia. *Aphasiology*. 2006;20(02–04):257–268.
65. Smania N, Aglioti S, Girardi F, et al. Rehabilitation of limb apraxia improves daily life activities in patients with stroke. *Neurology*. 2006;67(11):2050–2052.
66. MacDonald S, Wiseman-Hakes C. Knowledge translation in ABI rehabilitation: a model for consolidating and applying the BRAIN INJURY 57 evidence for cognitive-communication interventions. *Brain Inj*. 2010;24(3):486–508.
67. Dahlberg CA, Cusick CP, Hawley LA, et al. Treatment efficacy of social communication skills training after traumatic brain injury: a randomized treatment and deferred treatment controlled trial. *Arch Phys Med Rehabil.* 2007;88(12):1561–1573.
68. McDonald S, Tate R, Togher L, et al. Social skills treatment for people with severe, chronic acquired brain injuries: a multicenter trial. *Arch Phys Med Rehabil.* 2008;89(9):1648–1659.
69. Halbauer JD, Ashford JW, Zeitzer JM, Adamson MM, Lew HL. Neuropsychiatric diagnosis and management of chronic sequelae of war-related mild to moderate traumatic brain injury. *J Rehabil Res Dev*. 2009;46(6).
70. Cicerone KD, Langenbahn DM, Braden C, et al. Evidence-based cognitive rehabilitation: updated review of the literature from 2003 through 2008. *Arch Phys Med Rehabil.* 2011;92(4):519–530.
71. Togher L, Wiseman-Hakes C, Douglas J, et al. INCOG recommendations for management of cognition following traumatic brain injury, Part IV: cognitive communication. *J Head Trauma Rehabil*. 2014;29(4):353–368.
72. Rietdijk R, Power E, Attard M, Heard R, Togher L. Improved conversation outcomes after social communication skills training for people with traumatic brain injury and their communication partners: a clinical trial investigating in-person and telehealth delivery. *J Speech Lang Hear Res*. 2020;63:615–632.
73. Dou Z, Man D, Ou H, Zheng J, Tam S. Computerized errorless learning-based memory rehabilitation for Chinese patients with brain injury: a preliminary quasi-experimental clinical design study. *Brain Inj*. 2006;20(3):219–225.
74. Clare L, Jones RS. Errorless learning in the rehabilitation of memory impairment: a critical review. *Neuropsychol Rev*. 2008;18(1):1–23.
75. Taverni GS, Litchtman SW, Joseph P. Donepezil mediated memory improvement in traumatic brain injury during post acute rehabilitation. *Brain Inj*. 1998;12(1):77–80.
76. Zhang L, Plotkin RC, Wang G, Sandel ME, Lee S. Cholinergic augmentation with donepezil enhances recovery in short-term memory and sustained attention after traumatic brain injury. *Arch Phys Med Rehabil*. 2004;85(7):1050–1055.
77. Silver J, Koumaras B, Chen M, et al. Effects of rivastigmine on cognitive function in patients with traumatic brain injury. *Neurology*. 2006;67(5):748–755.
78. Wong G, Wong R, Mok V, et al. Rivastigmine for cognitive impairment after spontaneous subarachnoid haemorrhage: a pilot study. *J Clin Pharm Therapeut.* 2009;34(6):657–663.

Abbreviations list

SAH: Subarachnoid hemorrhage
aSAH: aneurysmal subarachnoid hemorrhage
TBI: Traumatic brain injury
ADL: Activity of daily living
FIM: Functional independence measure
DRS: Disability rating scale
APT: Attention processing training
Problem-solving training: (PST)
CIAT: Constraint-induced aphasia therapy